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suspected occupational dermatitis should be seen while the eruption is still in its early stages by a small group consisting of a general practitioner (the family doctor or examining medical practitioner or both), a dermatologist, and an industrial medical officer. The same group would also be available to cover, if necessary, "the long period of observation" referred to by Bettley.

It is always difficult to alter any system which has so many vested interests, but if the profession was supported in its desire to improve matters by the Ministry of Pensions and National Insurance this would at least be a start. There would appear to be support from trade unions for a change in the present system of assessment.[2] Despite the many practical difficulties of group assessment, if it received the support of all interested parties and its gradual development were accepted, it would seem to offer many advantages to both the industrial worker and the Ministries responsible for his welfare. The group approach, if developed, would allow certification to take place as an aspect of medical care along with treatment, investigation, and rehabilitation. It is surely time now for all concerned in the assessment of occupational dermatitis, both lay and medical, to discuss frankly the best methods of bringing to the worker in industry the expert facilities available within the framework of the National Health Service.

Self-poisoning

The steep rise in the consumption of barbiturates has for some time given cause for serious concern. It indicates there is something wrong with the practice of medicine. If, as M. Balint[1] says, the patient takes his doctor as a drug, the excessive demand for drugs reflects the doctor's inability to give enough of himself. The further increase in general prescribing after the abolition of the prescription charge suggests that doctors are in danger of losing control of the situation. The quantity of barbiturates prescribed in this country doubled between 1953 and 1959.[2] It might have doubled again since. The number of suicides attributable to barbiturate poisoning rose from 104 during 1941–50 to 735 during 1958–62. Professor Neil Kessel, in his Milroy Lectures published in this and last week's issues of the *B.M.J.*, warns against excessive prescribing of narcotic drugs. He is highly critical of the ease with which aspirin can be obtained in Great Britain, for there is hardly any shop or public place where it cannot be bought in unlimited quantities. The lesson of his studies is that unless doctors exercise restraint in the prescribing of hypnotics and unless the availability of salicylates is brought under some control all efforts towards a reduction of the suicide rate will be nullified.

Self-poisoning, says Professor Kessel, is "a fashion that has so developed over the last 20 years that to-day we regard it almost as commonplace," but this needs to be seen in historical perspective. Self-poisoning was heading the list of methods of suicidal acts long before barbiturates and other sedatives became easily available. F. Hopkins,[3] F. C. Lendrum,[4] and G. Donalies[5] reported on large series of attempted suicides admitted to hospital at Liverpool, Detroit, and Berlin between 1923 and 1935. The large majority had taken some kind of poison, many of them corrosive liquids or iodine. Only a small fraction had used narcotics in those days. Even at the beginning of this century self-poisoning was common. A considerable proportion of patients of all types admitted to the Charité in Berlin after suicidal attempts between 1892 and 1905[6] had taken poison, most of them corrosive liquids or other noxious chemicals.

It is not self-poisoning that is the new fashion to-day but the extensive use of certain drugs, especially of the barbiturates, which have ousted almost all the poisons used previously. Nor in the days before the barbiturates did the majority of people who took poison die of it. There always has been a large proportion of cases in which the ingestion of poison was relatively harmless to life. However, many of the older doctors practising to-day will remember the terrible damage sometimes caused by small amounts of corrosive liquids. It is a blessing that the barbiturates have no such effects.

Many acts of self-damage tend to be regarded as purely demonstrative and manipulative, especially when the patients deny suicidal intent. J. E. Lennard-Jones and R. Asher[7] called them "pseudocides," an unfortunate term because it could be understood to include pseudohomicide as well as pseudosuicide. "Pseudocides" are people who do not really want to die. Professor Kessel, too, divides his cases into those who want to die and those who want to live. He believes that four-fifths of his cases belong to the latter group. This assessment is based on the ineffectiveness of the poison taken or on the circumstances of the act. But could the patients know that they took no risk and that they were certain to survive?

The literature on suicidal acts has been bedevilled by semantic confusion. Until recently the term suicide was often used for both the fatal and the non-fatal act. The necessity to distinguish between the two has in recent years been accepted. Now Professor Kessel proposes to drop "attempted suicide" and speak of "self-poisoning" or "self-injury" instead. But "self-poisoning" is apt to lead to new confusion because most people when told that a person has poisoned himself will think that he is dead. Certainly "attempted suicide" is not a good term either, for it sounds too rational and suggests a planned effort, but it does refer to the self-destructive component of the act and the risk the individual runs in taking a poison the effect of which he is unable to judge. Though most of the acts of "attempted suicide" are impulsive, as Professor Kessel points out, those persons have almost invariably been preoccupied with death and suicide previously. Moreover, they usually show a complex behaviour pattern. E. Stengel[8 9] has pointed out that many suicidal attempts and some suicides are committed in the mood "I don't care whether I live or die" rather than with a clear and unambiguous determination to end life. Most people are ambivalent and muddled in their intentions at the time of the act, however much or little they may have thought of suicide before. They want to live and to die at the same time, usually the one more than the other, and do not lend themselves to clear classification. Uncertainty of outcome, as seen by the patient and not by the doctor, is a feature of most suicidal acts. These have been aptly described

1 Balint, M., *The Doctor, His Patient and the Illness*. London. 1957.
2 Brooke, E. M., and Glatt, M. M., *Med. Sci. Law*, 1964, **4**, 277.
3 Hopkins, F., *J. ment. Sci.*, 1937, **83**, 71
4 Lendrum, F. C., *Amer. J. Psychiat.*, 1933, **90**, 479.
5 Donalies, G., *Mschr. Psychiat. Neurol.*, 1928. **69**, 380.
6 Stelzner, H., *Analyse von 200 Selbstmordfällen*. Berlin. 1906.
7 Lennard-Jones, J. E., and Asher, R., *Lancet*, 1959, **1**, 1138.
8 Stengel, E., *Comprehens. Psychiat.*, 1960, **1**, 71.
9 —— *Lancet*, 1963, **1**, 233.
10 —— *Proc. roy. Soc. Med.*, 1952, **45**, 613.

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as Janus-faced, looking one way towards death and the other towards life.[10] Survival depends on the relationship between the self-destructive and the life-preserving tendencies. If the former are entirely absent we are not dealing with a suicidal attempt.

Steroid Glaucoma

In 1954 J. François[1] pointed out that cortisone drops applied to the eye for the treatment of conjunctivitis increased the intraocular pressure, reduced visual function, and caused optic atrophy. H. Goldmann[2] brought further cases to our notice in 1962. Unfortunately the problem is still with us.

At a recent meeting of the Ophthalmological Society of the United Kingdom S. J. H. Miller[3] described six cases of corticosteroid-induced glaucoma, and five of these patients had severe loss of visual field which was permanent. The primary condition for which each patient was treated was episcleritis, atopic eczema, allergic conjunctivitis, pinguecula, vernal catarrh, and sympathetic ophthalmitis. None had a family history of glaucoma so far as was known. All of them complained of intermittent haloes or blurred vision and had considerably raised intraocular pressure. They responded dramatically to cessation of therapy in that the intraocular pressure returned to normal limits with one exception. None of them showed continuing deterioration of the field of vision after cessation of treatment, and indeed one or two showed an improvement; the severity of the field loss was proportional to the total quantity of steroid given.

In recent years the effects of topical corticosteroids on the fluid dynamics of the eye have received careful study, chiefly by B. Becker[4 5] and M. F. Armaly.[6–9] The topical corticosteroids which have been reported to increase the pressure are cortisone itself, hydrocortisone, betamethasone, and dexamethasone in ascending order of potency. The hypertensive response over a period of four weeks is always greater in eyes with open-angle glaucoma. The rise is not accompanied by signs of congestion and resembles that seen clinically in glaucoma simplex. It has also been found that an individual with apparently normal eyes who has a parent with open-angle glaucoma reacts to topical corticosteroids by a rise in pressure greater than that of his contemporary who has no family history, and the rise is comparable in size to that found in a patient with frank glaucoma.

Becker[5] proposed a genetic hypothesis to explain the difference in response to topical corticosteroids found in a normal eye and in one with open-angle glaucoma. Glaucoma simplex is considered to be a heritable trait and to represent the homozygous recessive state with a frequency of 4% in the general population. Theoretically the heterozygous state should have a frequency of 32% and the homozygous dominant 64%. The size of the steroid effect on intraocular pressure divides a series of normal people into two groups: group I with the less response consists of individuals who do not bear the recessive gene, and group II with the greater response consists of individuals having the recessive gene either in the heterozygous or in the homozygous state.

Armaly[9] has carried this one stage further. Applying 0.1% dexamethasone 21-phosphate three times daily to the right eye in 80 volunteer subjects, he has defined three significantly different groups by the size of the rise in pressure. Group I showed a low level of response with an average rise of 1.6 mm. and formed 66.2% of the sample. Group IIa showed an intermediate level of response with an average rise of 10 mm. Hg; this formed 28.8% of the sample. Group IIb showed a rise of pressure of 16 mm. Hg or greater, and this group formed 5% of the sample. These percentages are very close to the theoretically derived figures based on Becker's genetic theory.

G. Paterson[10] has reported a study on a series of siblings and children of patients known to have glaucoma simplex. The results were analysed so as to compare Armaly's figures from a sample of the general population with those taken from a group of people who were known to carry the gene of glaucoma. Theoretically none of these latter patients should be in group I, but should show either an intermediate rise indicating the heterozygous state or a large rise indicating the preclinical stage of glaucoma, the disease being age-dependent. In fact Paterson's figures showed that 33% of the siblings and 33% of the children actually fitted into group I.

There are several possible explanations for this divergence from expectation. The total dosage of steroids employed by the two workers was not the same. It may be that the penetrance of the gene is incomplete in some cases or that the heritable trait is not the disease of glaucoma itself but rather the pressure response to corticosteroids. Further investigation is required to resolve this paradox.

Meanwhile the clinical lesson to be learnt from this work is the danger of topical corticosteroids, particularly beta- and dexamethasone, when prescribed for a period longer than a week unless ophthalmic supervision is close. The possibility, however, of using corticosteroid eye drops to detect genetic liability to glaucoma may prove to be of practical value.

Electroencephalography in Childhood

The sudden occurrence of a local lesion in the brain is usually accompanied by a prompt change in the electrical activity of the neighbouring regions. This can be recorded by the electroencephalograph (E.E.G.), though the relation between the lesion of the brain and the concomitant electrical changes is not fully understood. Frequently, however, the E.E.G. record changes continually until the condition has either resolved or become static.[1]

The E.E.G. findings in childhood differ from those in adults, both in normal subjects and in patients with diseases of the brain. For example, the progression of brain tumours in children is considerably different from that of adults, both clinically and electroencephalographically. Similarly, acute lesions of the brain, such as abscesses, various forms of encephalitis, or vascular disorders, also have different features according to the age of the patient. In children the E.E.G. changes are often disproportionately greater than those seen in apparently similar conditions in adults.

[1] François, J., *Ann. Oculist (Paris)*, 1954, **187**, 805.
[2] Goldman, H., *Arch Ophthal.*, 1962, **68**, 621.
[3] Miller, S. J. H., *Trans. ophthal. Soc. U.K.*, 1965, in the press.
[4] Becker, B., and Mills, D. W., *Arch. ophthal.*, 1963, **70**, 500.
[5] —— and Hahn, K. A., *Amer. J. Ophthal.*, 1964, **57**, 543.
[6] Armaly, M. F., *Arch. Ophthal.*, 1963, **70**, 482.
[7] —— ibid., 1963, **70**, 492.
[8] —— ibid., 1964, **71**, 636.
[9] —— Gilston Glaucoma Symposium, 1965, in the press.
[10] Paterson, G., *Trans. ophthal. Soc. U.K.*, 1965, in the press.

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